We’ve spent billions of dollars fighting it but has cancer been fundamentally misunderstood?

Ever since Richard Nixon officially declared a war on cancer in 1971 through the signing of the National Cancer Act, more than $100 billion in taxpayer money has been spent on research and drug development in an attempt to eradicate the disease. Trillions more have been spent by the cancer patients themselves, but with disappointing results.
Even after four decades of waging full-scale war on cancer, both conventional (surgery and chemotherapy) and nuclear (radiotherapy), one in every four Americans will be diagnosed with the disease during their lifetimes. This number is projected to grow, unabated, not unlike the process of cancer itself.

Could this colossal failure reflect how profoundly misunderstood the condition is, and how misguided our attempts to prevent and treat it?

What Is Cancer?

Perhaps we need to return back to the fundamental question, “What is cancer?” After all, until we find an accurate definition, all attempts to prevent and treat a disease we do not understand are doomed to fail.

For the past half century, the “mutational theory” has provided the prevailing explanation for the cause of cancer. As the story goes, accumulated mutations within our cells lead a few susceptible ones to “go berserk,” their “insane” and “violent” behavior a result of multiple destructive events to the intelligent code within the cell (DNA) that normally keeps them acting in a “civilized” manner relative to the larger multicellular community as a whole (i.e. the body). In this view, these rogue cells replicate incessantly and form a tumor which spreads outward, in many ways simulating the characteristics of an infectious process within the host, until the growths obstruct vital processes, resulting in morbidity and death.

According to this theory, which was heavily influenced by the Darwinian theory of evolution and is sometimes called “internal Darwinism,” what drives the evolution of the healthy cells into cancerous ones is a process very similar to natural selection. Random mutations beneficial to the survival and reproduction of cancerous cells in a tumor are naturally selected for and conserved, driving them toward malignancy. Damage to the DNA can occur either through inheriting defective DNA sequences (“bad genes” in the family) or exposures to DNA damaging chemicals (tobacco, for example) or radiation.
While this view has some explanatory value, it can also be quite misleading. For instance, a fundamental tenet of evolution is that random mutations are almost always harmful, resulting in immediate cell death. Cancer cells, however, seem to get quite “lucky,” because they appear to thrive on them. Rather than dying like normal cells when faced with random mutations, they exhibit the exact opposite response: They become immortalized, incapable of undergoing the programmed cell death required of healthy cells.

Is randomness and chaos, then, really at the root of the transformation of healthy cells into cancer?

Tumors—collections of cancer cells—express highly organized behaviors, after all, which are seemingly impossible to induce through strictly random forces such as mutation. They are capable of building their own blood supply (angiogenesis); of defending themselves by silencing cancer-suppression genes and activating tumor-promoter genes; of secreting corrosive enzymes to move freely throughout the body; of altering their metabolism to live in low-oxygen, high-sugar and acidic environments; and of removing their own surface-receptor proteins to escape detection by white blood cells. Could these complex behaviors really be a result of random mutations? And is it possible that random mutations could result in the formation of the same “lucky” set of genetic properties, each and every time a new cancer forms in a human?

Random mutations, no doubt, play a major role in the initiation and promotion of cancer, but are not alone sufficient for a complete explanation. One group of scientists, in fact, have offered a much more compelling explanation, suggesting that multiple mutations reveal an ancient survival program within the cell.

A Prehistoric Defense Mechanism

A brilliant new theory, introduced by Arizona State University scientist Paul Davies and Australian National University scientist Charles Lineweaver, sheds much needed light on the true nature of cancer. According to Davies, “Cancer is not a random bunch of selfish rogue cells behaving badly, but a highly-efficient pre-programmed response to stress, honed by a long period of evolution.”
In their seminal paper, titled “Cancer tumors as Metazoa 1.0: Tapping genes of ancient ancestors,” Davies and Lineweaver propose that cancer is an evolutionary throwback, drawing from a genetic “toolkit” at least a billion years old, and which still lies buried—normally dormant—deep within the genome of our cells. Davies calls this subterranean genetic layer “Metazoa 1.0,” and it contains pathways and programs that were once indispensable for our ancient cellular predecessors and their early proto-communities to survive in a radically different environment.

Without the highly differentiated cells and specialized organs of higher multicellular/animal life (what Davies calls “Metazoa 2.0”), cells with the genetics of Metazoa 1.0 would have favored traits that enabled them to survive direct contact with what was a much different and harsher (to us) environment.

For example, 1 billion years ago atmospheric oxygen was exceptionally low, since photosynthesis has not yet evolved to produce an abundant supply. This means that cellular life at that time would have had to learn to thrive in a low- or no-oxygen environment, which is exactly what cancer cells do, using aerobic glycolysis for energy instead of oxidative phosphorylation.

Davies and Lineweaver summarize their view as follows:

“The genes of cellular cooperation that evolved with multicellularity [animal life] about a billion years ago are the same genes that malfunction to cause cancer. We hypothesize that cancer is an atavistic condition that occurs when genetic or epigenetic malfunction unlocks an ancient ‘toolkit’ of pre-existing adaptations, re-establishing the dominance of an earlier layer of genes that controlled loose-knit colonies of only partially differentiated cells, similar to tumors. The existence of such a toolkit implies that the progress of the neoplasm [cancer] in the host organism differs distinctively from normal Darwinian evolution.”

Instead of viewing the hallmark trait of cancer, namely incessant proliferation, as a newly evolved trait spurned by random mutations, it would be considered the default state of the cell, having been developed a billion years ago when “not dying” would have been the first priority. Remember, this ancestral assemblage of cells would not have had the differentiation of cell type and specialization of tissue associated with higher animals—skin, hair, claws, etc.—with which to protect itself against the environment.
Damage to the skin in animals, for instance, results in the rapid death and sloughing off these “extra” cells, to be replaced by new healthy ones. A still barely multicellular entity would not have this luxury, and would entrench itself within genetic traits associated with resilience—the ability to resist all manner of environmental assault—and would express a highly “selfish” form of behavior we now consider a fundamental property of cancer.

If cancer is an ancient survival program unmasked, this does not mean that the “mutation theory” does not still hold some truth. Genetic damage and mutations do in fact contribute to cancer, but rather than view them as causing the complex set of behaviors associated with cancer, they unmask an atavism, an already existent set of genetic programs. (See sidebar, bottom of page.) For instance, we know of more than 100 oncogenes that exist within our DNA which we share with a vast array of different species, including the fruit fly. This indicates how ancient (at least 600 million years old) and universal they are (found in most multicellular organisms).

Numerous studies confirm that dinosaurs had tumors. These cancer-promoting genes are normally suppressed by more recently evolved genes (Metazoa 2.0), such as tumor-suppressor genes, but when enough damage to the more recently evolved genetic overlay occurs, the system goes into “safe mode” and the older genetic pathways (Metazoa 1.0) are activated once more.

Within the horizon of this new way of thinking, cancer can no longer be viewed as some predestined genetic time bomb setting itself off within us, nor simply a byproduct of cumulative exposures to genotoxic substances, alone. Rather, cancer is an ancient survival response to an increasingly toxic environment, an increasingly unnatural diet and compromised immune function. These cells have learned to survive the constant abuse, and have flipped into survival mode, which is self-centered, hyper-proliferative (constant selfrepair/ replication) and aggressive (metastatic). In other words, what does not kill them makes them stronger.

Cancer as Survival Tactic

Cancer can no longer be viewed as something bad that happens to an intrinsically healthy body. Rather, cancer is something the body actively does in response to an intrinsically unhealthy cellular, bodily and planetary environment. Instead of an expression of bodily deviance, it may be expressive of bodily intelligence, and the capability of our cells to survive in conditions that otherwise threaten to destroy cells.
This perspective also sheds much-needed light on the devastating nature of chemotherapy and radiotherapy. Tumors contain a broad range of cells, many of which are intrinsically benign (will never become malignant or cause damage to the organism) and some of which keep more malignant populations in check.

The invasive cells are more primordial in their genetic configuration (Metazoa 1.0) due to just how much shock/damage/poisoning they have been made to endure during their life cycles. It is exactly these cells, therefore, that are most resistant to the chemo, and less likely to die when exposed to it. The chemotherapy and radiation, therefore, actually kill the very cells that do not represent a threat, and select for more invasive ones.

This explains why at first the introduction of chemotherapy/ radiation may cause tumor regression, but the small population that survives (including cancer stem cells) technically comes back even stronger thereafter. In the same way that antibiotics like methicillin spawned the monster that is methicillin-resistant Staphylococcus aureus, which creates a population of bacteria with highly up-regulated multidrug-resistant proteins and genes, chemotherapy and radiation create a genetically more-resistant population of super-cancers, and often are the reason why the patient dies. Sadly, in these cases the death is blamed on the “chemoresistant” and “radioresistant” cancer and the victim is killed by the very treatment they were told they would die much sooner without.

Cancer Is a Symptom

So, instead of a monolithic “disease,” it makes more sense to view cancer as a symptom of cellular and environmental conditions gone awry; in other words, the environment of the cell has become inhospitable to normal cell function, and in order to survive, the cell undergoes profound genetic changes, drawing ancient genetic pathways which we associate with the cancerous personality (phenotype). This “ecological” view puts the focus back on the preventable and treatable causes of the “disease,” rather on some vague and outdated concept of “defective genes” beyond our ability to influence directly.

It also explains how the “disease” process may conceal an inherent logic, if not also a healing impulse, insofar as it is an attempt of the body to find balance and survive in inherently unbalanced and dangerous conditions. Fundamentally, we need to shift our thinking away from
the view that cancer is something unnatural that happens to us, to one where we see that cancer is something natural our body does to survive unnatural conditions. Change and improve those conditions, and you do more to change cancer than attacking it as if you were fighting a war.

Side Bar:

Cancer as Atavism

The concept of cancer-as-atavism can be explained this way: An atavism is an older genetic trait that is no longer used, and therefore suppressed by newly evolved genes. An example is webbed feet in humans. Everyone in the womb has them, but as embryogenesis proceeds, genetic sequences kick in that cause them to disappear. This is done through a process of “programmed cell death,” also known as apoptosis. The body simply turns on the apoptosis genes in the tissue associated with webbing between the toes, and those cells peacefully disassemble themselves, resulting in normal, web-free hands and feet. The interesting thing is that cancer cells are cancerous because they do not die. They have either forgotten how to undergo programmed cell death (apoptosis), or, have been forced through injury (genetic damage) or environmental pressures (epigenetic changes) to suppress the genes that enable them to die. Cancer cells, in effect, draw from an ancient genetic tool kit which its predecessors more than a billion years ago used to survive what was at the time a very harsh environment. Cells had not yet formed the highly evolved multicellular communities found in animals, and the replicating trait was preferable to dying.